the activating interface[15,16]. This interface is formed largely by a four-helix bundle that includes helices α13 and α14 of the TIM barrel (together with their 2-fold-related symmetry mates) (Fig. 2*b*). The complementation peptide contributes to the interface in two different ways: first, it participates directly in the interfacial contact region; second, it helps indirectly by stabilizing the four-helix bundle.

Different β-galactosidases (Fig. 3) have poor sequence conservation in the vicinity of both the long and the activating interfaces. In particular, the Sth, Lbu and CbgA sequences have a 10–12-residue deletion within the donated loop, and insertions in the vicinities of residues 420 and 493. These changes near the active-site region and the four-helix bundle suggest that the Sth, Lbu and CbgA may lack an activating interface or that the active site might be formed within a single monomeric chain. Consistent with this, Lbu β-galactosidase appears to be dimeric[18].

Why is *E. coli* β-galactosidase so large? Lysozymes cleave the same glycosidic bond but are ~1/20th the size. The natural substrates for lysozyme, bacterial cell walls, are highly crosslinked peptidoglycans, so perhaps lysozyme needs to be small to access susceptible bonds[19]; β-galactosidase is subject to no such restriction. Inspection of the structure suggests that the different parts combine together in an interdependent way to form the active enzyme. Different regions contribute to the active site, to the long interface, or to the activating interface (Fig. 3). There is no large segment that could be removed without having a deleterious effect. It is conceivable that the protein arose from different structural domains whose combined activity happened to be useful to the cell, and became trapped in sequence space at roughly its present size. □

Received 5 April; accepted 20 May 1994.

1. Jacob, F. & Monod, J. *J. molec. Biol.* **3,** 318–356 (1961).
2. Fowler, A. & Zabin, I. *J. biol. Chem.* **253,** 5521–5525 (1978).
3. Kalnins, A., Otto, K., Ruther, U. & Müller-Hill, B. *EMBO J.* **2,** 593–597 (1983).
4. Jacobson, R. H. & Matthews, B. W. *J. molec. Biol.* **223,** 1177–1182 (1992).
5. Tronrud, D. E., Ten Eyck, L. F. & Matthews, B. W. *Acta crystallogr.* A**43,** 489–501 (1987).
6. Holmgren, A. & Brändén, C. I. *Nature* **342,** 248–251 (1989).
7. Brändén, C. Q. *Rev. Biophys.* **13,** 317–330 (1980).
8. Matthews, B. W., Jansonius, J. N., Colman, P. M., Schoenborn, B. P. & Dupourque, D. *Nature New Biol.* **238,** 37–41 (1972).
9. Celada, F., Ullmann, A. & Monod, J. *Biochemistry* **13,** 5543–5547 (1974).
10. Ring, M. & Huber, R. E. *Arch. Biochem. Biophys.* **283,** 342–350 (1990).
11. Gebler, J. C., Aebersold, R. & Withers, S. G. *J. biol. Chem.* **267,** 11126–11130 (1992).
12. Cupples, C. G., Miller, J. H. & Huber, R. E. *J. biol. Chem.* **265,** 5512–5518 (1990).
13. Ullmann, A., Jacob, F. & Monod, J. *J. molec. Biol.* **24,** 339–343 (1967).
14. Langley, K. E., Villarejo, M. R., Fowler, A. V., Zamenhof, P. J. & Zabin, I. *Proc. natn. Acad. Sci. U.S.A.* **72,** 1254–1257 (1975).
15. Weinstock, G. M., Berman, M. L. & Silhavy, T. J. in *Gene Amplification and Analysis* (eds Papes, T. S., Rosenberg, M. & Chirikjian, J. G.) 27–64 (Elsevier, New York, 1983).
16. Zabin, I. *Molec. cell. Biochem.* **49,** 87–96 (1982).
17. Henderson, D. R., Friedman, S. B., Harris, J. D., Manning, W. B. & Zoccoli, M. A. *Clin. Chem.* **32,** 1637–1641 (1986).
18. Adams, R. M. *et al. J. biol. Chem.* **269,** 5666–5672 (1994).
19. Heinz, D. W. & Matthews, B. W. *Prot. Engng* **7,** 301–307 (1994).
20. Sakabe, N. *Nucl. Inst. Meth.* A**303,** 448–463 (1991).
21. Higashi, T. *J. appl. Cryst.* **22,** 9–18 (1989).
22. Terwilliger, T. & Eisenberg, D. *Acta crystallogr.* A**39,** 813–817 (1983).
23. Rayment, I. *Acta crystallogr.* A**39,** 102–116 (1983).
24. Kraulis, P. J. *J. appl. Cryst.* **24,** 946–950 (1991).
25. Kabsch, W. & Sander, C. *Biopolymers* **22,** 2577–2637 (1983).
26. Stokes, H. W., Betts, P. W. & Hall, B. G. *Molec. Biol. Evol.* **2,** 469–477 (1985).
27. Buvinger, W. E. & Riley, M. *J. Bact.* **163,** 850–957 (1985).
28. Schroeder, C. J., Robert, C., Lenzen, G., McKay, L. L. & Mercenier, A. *J. gen. Microbiol.* **137,** 369–380 (1991).
29. Schmidt, B. F., Adams, R. M., Requadt, C., Power, S. & Mainzer, S. E. *J. Bact.* **171,** 625–635 (1989).
30. Hancock, K. R. *et al. J. Bact.* **173,** 3084–3095 (1991).
31. Gaykema, W. P. J. *et al. Nature* **309,** 23–29 (1984).
32. Rossmann, M. G. *et al. Nature* **317,** 145–153 (1985).

ACKNOWLEDGEMENTS. We thank S. Clarke and M. Krevolin for cell fermentation; J. A. Wozniak and S. Snow for assistance with protein purification and crystallization; A. Nakagawa and N. Sakabe for their help at the Photon Factory; R. Albright, E. P. Baldwin, M. Blaber, S. L. Roderick, W. Tulip, L. H. Weaver and K. P. Wilson for help with data collection; S. L. Roderick, I. Rayment and M. G. Rossmann for technical advice; D. E. Tronrud for modifications to TNT; B. Branchaud and L. Blaszczak for synthesis of mercurated-c-galactose; L. Holm for database searches; and S. G. Withers, J. H. Miller, B. F. Schmidt and I. Zabin for discussion. This work was supported in part by a grant from the NIH to B.W.M.

CORRECTION

A map of a collisionally evolving dust disk around Fomalhaut

S. Alan Stern, Michel C. Festou & David A. Weintraub

Nature **368,** 312–314 (1994)

WE recently reported[1] mapping observations made in February 1993 indicating a detection of extended 1.3-mm emission around the nearby, main-sequence infrared excess star Fomalhaut. On 17 and 18 April 1994 we made similar but more sensitive observations of Fomalhaut at the same observatory (IRAM). These observations revealed that the extended emission we earlier reported appears to have been an artefact unique to the 1993 data. We therefore reprocessed the 1993 maps with improved facility software (R. Zylka, personal communication), and found that in each of the three 1993 maps there was a "source" near Fomalhaut, but that the precise position of this "source" changes by several arcsec from map to map. Thus, these sources cannot be real, but are probably due to variations in the thermal foreground to be of comparable size and brightness to a plausible source around the star. Apparently, the sum of the "sources" in the 1993 maps produced an extended object, fortuitously placed signal at the position of Fomalhaut on the sky.

On 18 and 19 April 1994 we made even more sensitive observations at IRAM using the same instrument in on–off (rather than mapping) mode; we used a chop-throw of 45 arcsec. These data reveal the detection of an unresolved, central source at a level of 7.1 ± 3.1 mJy, and a 3σ upper limit for a total flux of 51 mJy in the six, 20 arcsec-diameter channels that surround the central channel. This revised flux density contradicts our previous report[1] on the size, brightness, and physical characteristics of the Fomalhaut disk, and is consistent with results described in Scientific Correspondence on page 766 of this issue by Chini *et al.*[2]. A detailed study of the ambiguities and past contradictions in millimetre-wave observations of very faint sources is reported in ref. 3. □

1. Stern, S. A., Festou, M. C. & Weintraub, D. A. *Nature* **368,** 312–314 (1994).
2. Chini, R., Krugel, E., Kreysa, E. & Zylka, R. *Nature* **369,** 766 (1994).
3. Weintraub, D. A. & Stern, S. A. *Astr. J.* (in the press).